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Job strain and clinical depression – Authors' reply

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Mikkelsen and colleagues (2017) disagree with our conclusion that "Job strain may precipitate clinical depression among employees" (Madsen et al. 2017). In response, we would like to remind the reader that the individual participant data (IPD) meta-analysis part of our paper was based on a pre-planned study protocol to test a specific hypothesis (Madsen et al. 2014). To minimize researcher bias such as p-hacking and post-hoc decision-making we reported the aims, exposure and outcome definitions, analysis protocol and decision to publish the results before starting the analyses (Madsen et al. 2014). This type of study registration has long been the standard in randomized controlled trials, although very few reports on cohort studies to date have followed this approach to strengthen evidence (Kivimäki et al. 2013).

The purpose of our project was to test the hypothesis that individuals experiencing job strain are more likely to develop clinical depression than individuals without job strain. Our meta-analysis of the published literature on job strain and depression risk, assessed following a clinical diagnostic interview (the gold-standard method in research studies), yielded a pooled odds ratio of 1.77 (95% CI 1.47-2.13) among participants who were free of clinical depression at baseline. The IPD meta-analysis of 14 unpublished studies on job strain and the risk of hospital-treated clinical depression yielded a pooled hazard ratio of 1.27 (95%CI 1.04-1.55) among participants with no previous history of hospital treatment for clinical depression. We interpreted this as supportive evidence for the hypothesis. Several supplementary analyses corroborated this conclusion, including an analysis showing a dose-response association between exposure to job strain at multiple time-points and the risk of clinical depression (Madsen et al. 2017, Supplementary Table S5). The association of job strain with hospital treatment for depression disappeared when we adjusted for (non-clinical) depressive symptoms at baseline, but further analysis indicated that previous exposure to job strain may have caused some of the non-clinical depressive symptoms. Taking the results from both the hypothesis-testing and the supplementary analyses together we stand by our conclusion that "job strain may precipitate clinical depression among employees".

Mikkelsen and colleagues criticize us for examining the statistical interaction of demands and control only in a supplementary analysis and not in the main analyses. We did this because we did not hold

that, as Mikkelsen and colleagues argue, the (additive or multiplicative) interaction of demands and control is "the hallmark of Karasek's job strain theory". As Stan Kasl, who critically followed the job-strain model for decades, noted, "the precise nature of the interaction has never been fully spelled out" by Karasek and his colleagues (Kasl 1996, p. 48). De Jonge and Kompier (1997, p. 245) echoed this sentiment, concluding that "the phenomenon of 'interaction' is not clearly defined in the model". Consequently, and in accordance with the overwhelming majority of literature on job strain, we did not calculate additive or multiplicative interaction terms in our main analysis, but used a dichotomous operationalization (being versus not being exposed to job strain), and applied a quadrant definition (including the categories high job strain, low job strain, passive job and active job) in the sensitivity analysis. This was not a post-hoc decision: we chose and documented these operationalizations before linking the exposure and the endpoint data (Fransson et al. 2012, Madsen et al 2014). Indeed, there are further examples indicating that methods other than interaction terms may be more meaningful in analyses of the interplay between two variables. One well-known example from epidemiology is the body mass index, which combines height and weight into a sensitive indicator of cardio-metabolic risk (Wells 2014).

Mikkelsen and colleagues suggest that, in the light of our data, interventions to reduce the risk of clinical depression should focus on job control rather than job strain. Their interpretation of the evidence is not without problems. Making inferences about the effectiveness of interventions would require data on the degree to which job strain or job control are amenable to change: such data were not reported in our paper. Similarly, the question of which of the two components of job strain is more toxic or more amenable to intervention was not the focus of our pre-planned project. That would require a different approach addressing issues such as the extent to which reverse causation and covariates affect the associations of job demands and job control with clinical depression; whether an increase in job control is more strongly related to the reduction of depression risk than a decrease in job demands; and to what extent the benefits of changing either of the components differ between people with and without job

strain (i.e., those who have either low control or high demands but not both). We hope that our paper will motivate such research.

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